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CLINICAL NOTES ON APOPLEXY.

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In the remarks I have to make the term *apoplexy* will be used in its broadest sense, and as a general term will embrace not only the acute cerebral attack or "stroke" due to hemorrhage, but also that resulting from the plugging of a vessel. The apoplectic seizures occasionally met with in intracranial tumor or abscess, in general paralysis of the insane and in a few other conditions, will not be considered.

A "stroke" or an attack of apoplexy is vascular in origin and is due either to rupture of a blood vessel in the brain—cerebral hemorrhage—or to occlusion of a cerebral vessel—thrombosis or embolism. Hence the underlying pathology will be found in the circulatory system. With heart and blood vessels healthy a man is not stricken with apoplexy.

(1) *Cerebral hemorrhage* is due to rupture of a diseased blood-vessel in the brain, and, apart from traumatism, purpura and pernicious anæmia, probably never occurs with healthy vessels. As a result of chronic degeneration the walls of the cerebral arteries become weak and may partially give way at various points, forming minute miliary aneurisms. Able to withstand ordinary pressure, these little aneurisms are liable to rupture under excitement or sudden effort. In the great majority of cases there are associated with the arterial changes hypertrophy of the left ventricle and increased intra-arterial pressure as shown by the accentuated aortic second sound, and these changes are due to arteriosclerosis, either primary, or as an associated condition in chronic Bright's disease. In nearly all cases the rupture can be traced to three factors, viz: powerful left ventricle, high arterial pressure and weakened vessel wall. Cerebral hemorrhage is especially frequent between the ages of forty and sixty, that is, during the period when arterial degeneration is marked, while the power of the heart is still good. After sixty the heart becomes weaker, intra-arterial pressure is lower and rupture is progressively less frequent. As arterial degeneration is much more common in men than in women, so cerebral hemorrhage more frequently affects the former.

In about two-thirds of all cases the hemorrhage takes place in the internal capsule or *basal ganglia;* next most frequently in the cortex, and less often in the pons and cerebellum. When hemorrhage occurs in the brain tissue the blood tears and disorganizes the brain substance and soon forms a clot around which there is considerable œdema. This clot exerts sufficient pressure upon the adjacent brain tissue to impair or destroy its function, but the œdematous and compressed tissue will resume its function when the pressure is removed. Hence, the immediate and temporary effects of a cerebral hemorrhage are usually much more severe and extensive than the permanent ones, but as the absorption of the clot is slow the improvement in the symptoms is necessarily slow and gradual. As the œdema about a hemorrhagic lesion often subsides early there may be noticed a rapid improvement, which is, however, only partial and due to the relief of the pressure caused by the œdema. Of course, the fibers that have been destroyed and have degenerated never resume their function, hence in all cases where the hemorrhage has been at all extensive there will be some permanent defect.

(2) *In Cerebral Thrombosis* there is occlusion of an artery due to the gradual constriction formed by obliterating endarteritis or to the formation of a clot upon the roughened wall of the vessel. When the occlusion is complete nutrition is at once cut off from all the brain tissue supplied by the plugged vessel, and function is at once suspended in this area, but when the collateral circulation is established the function returns in all parts not permanently deprived of blood. Hence as in hemorrhage the immediate effects of the stroke may be extensive and severe, but in thrombosis the improvement may be so marked that but slight permanent defect remains. As the collateral circulation is usually established within a week the improvement which occurs in thrombosis or embolism is much more rapid than that seen in hemorrhage. Thrombosis is relatively much more common in the cortical arteries than in those of the base, hence the frequency of localized and incomplete palsies in this condition. In the cortex the collateral circulation is extensive, but in the arteries of the internal capsule and base it is very imperfect as the arteries are terminal. Hence there is much less recovery in lesions of the basal arteries than in those of the cortex. Sometimes the middle cerebral artery itself is plugged, in which case there will be wide-spread softening. When the clot forms in the vessel it extends backward to the next branch, and if a portion of it should be separated at this point it will be carried into this branch, plugging it and causing another attack with more extensive paralysis within the next day or two. Oedema rapidly appears in the brain territory supplied by the occluded vessel, but it subsides as the collateral circulation is established. The ultimate effect of thrombosis is softening from fatty degeneration in the area permanently deprived of blood with descending degeneration in all the fibers involved.

Thrombosis occurs in obliterating endarteritis from any cause. In the young, syphilis is responsible for the majority of cases of apoplexy; in the old, senile endarteritis is the underlying factor. In such cases slowing of the blood current from weakening of the heart may result in thrombosis, hence the stroke often appears during sleep or after exhaustion or fatigue.

(3) *In Cerebral Embolism* the artery, which is usually healthy is occluded by an embolus coming from the heart or large arteries, and is met with most frequently in cases of endocarditis in young persons. The pathological changes are exactly similar to those met with in thrombosis.

Though the actual pathological conditions that cause the apoplectic attack in hemorrhage, thrombosis and embolism are clear, well defined and distinct from one another, the immediate effects on the functions of the area of brain involved are so nearly alike that clinically it is often impossible to determine the exact nature of the lesion. Inasmuch as the treatment of a hemorrhagic attack is practically the opposite of that of the stroke of thrombosis, the differentiation of these two conditions is of the greatest importance. Unfortunately, however, it is impossible in many cases to determine whether the attack is due to hemorrhage or to occlusion of a vessel. In hemorrhage and embolism the attack is sudden because the accident to the vessel is of sudden occurrence. In thrombosis, though the narrowing of the vessel may have been gradual, the actual closure is usually quite sudden and the attack may be as rapid in its onset as in hemorrhage or embolism.

In differentiating the three conditions there are some features sufficiently characteristic as to possess considerable diagnostic significance though not one of them alone can be considered as conclusive. The chief points of diagnostic value are: age of patient, the existence of certain diseased conditions, exciting cause of an attack and time of occurrence, premonitory symptoms, suddenness of onset and nature of attack, unconsciousness, extent of paralysis and location of lesion, aphasia, reactionary fever, and rapidity and extent of recovery.

Age—Hemorrhage is most frequent between the ages of forty and sixty, thrombosis under forty and again in old age, and embolism in the young. The great majority of attacks of apoplexy occurring under forty and over sixty are thrombotic in origin.

Existence of Disease—The presence of arteriosclerosis, with cardiac hypertrophy, accentuated aortic second sound, high intra-arterial pressure and possible urinary evidences of chronic interstitial nephritis affords the conditions necessary for the occurrence of hemorrhage. Hence, if a man past middle life presents these signs and is suddenly stricken with apoplexy, we have strong presumptive evidence that there has been a rupture, especially if the attack occurred during excitement or physical exertion.

The existence of evidences of syphilis or of senile arterial degeneration, especially if associated with low blood pressure and weak heart or anæmia, would strongly favor thrombosis. Syphilitic endarteritis with thrombosis is undoubtedly the cause of the great majority of strokes under middle life.

A stroke in a young man or woman who presents signs of disease in the valvular region of the heart or in the aorte is probably embolic and this becomes almost a certainty if syphilis and Bright's disease can be excluded.

The exciting cause of an attack may be of distinct value when it can be determined. A stroke that occurs during the period of high arterial pressure and forcible heart action that are associated with mental excitement or unusual physical effort is very likely due to hemorrhage. It must not be forgotten, however, that rupture may take place when no such circulatory excitement is apparent. As the clotting in the vessel is favored by a feeble current, thrombosis is most likely to occur when the cardiac force is weak and the intra-arterial pressure low, hence the apoplexy of thrombosis frequently appears during sleep or during the exhaustion that follows fatigue, excitement or illness. The time of occurrence of embolism is probably of little, if any, value, in diagnosis.

Premonitory Symptoms—In *hemorrhage*, as a rule, there have been no premonitory symptoms of any value. Headache, vertigo, mental confusion and nausea are absent. There have been no slight attacks of vertigo or mental confusion followed by local paralysis and in nearly all cases the attack comes on suddenly without any warning. Likewise in *embolism*, premonitory symptoms are absent, though rarely a patient may have suffered from embolism in some other vessel, as for example, in the central artery of the retina producing sudden blindness, in the spleen or kidneys. It is especially in *thrombosis* that premonitory symptoms are common, though not always present. These prodromata may have extended back over weeks or months. The most significant symptoms are headaches, especially if induced by fatigue, mental or physical (in syphilitic cases the headache is usually worse towards night), attacks of vertigo coming on after effort or after a full meal, insomnia, mental confusion, loss of memory, numbness and paralysis. Numbness, if persistent or frequently recurring, and always referred to the same arm, leg or side, is a common symptom in these cases, and if associated with distinct signs of endarteritis is very significant, indicating as it does an abnormal state of the circulation in a localized area of the brain. The patient may have suffered from slight attacks of paralysis, localized to one arm, leg or one side of the face, or he may give a history of a temporary aphasia, followed later by paralysis in a limb. The paralysis in these cases is often slight; it may be discovered on rising in the morning, or it may appear after a few days of headache, vertigo or mental confusion. An attack of apoplexy in a man who gives such a history is almost surely due to thrombosis.

Mode of Onset—In hemorrhage and embolism the attack is so sudden in its onset that the patient often falls stricken to the ground, the paralysis being complete at once. The attack in thrombosis may be quite as sudden, but usually the patient is conscious of a numb or dead feeling in the affected limb and he may be confused or dazed for some minutes or even longer before the loss of power is observed. In some instances the loss of power at first is localized to part of one limb, then gradually extends to the whole limb and perhaps to the whole side, but some hours may elapse before the full extent of the paralysis is shown.

Loss of Consciousness is a most valuable symptom in differential diagnosis. In *hemorrhage* it is nearly always present, appears suddenly at the time of the attack, is usually complete and as a rule lasts for at least twenty-four hours, often much longer. In very slight hemorrhages the symptom may be absent, but when the lesion is sufficient to produce a complete though slight, hemiplegia, it is pretty certain to be present, and a marked hemiplegia from hemorrhage is, according to Russell, always attended by loss of consciousness at the time.

In the majority of cases of thrombosis there is no loss of consciousness, or if present it is incomplete and of short duration. The patient is, however, often dazed and confused for a few minutes or a few hours, but consciousness is not lost. His cerebration is slow and imperfect, but he can be partially roused and responds to questions or commands. Even in a complete hemiplegia from thrombosis, loss of consciousness is unusual and a complete hemiplegia, gradual or sudden in its onset, without coma means thrombosis.

In embolism consciousness is not usually lost. The coma that is seen in some cases is, as a rule, incomplete and of much shorter duration than that of hemorrhage and when recovered from, it passes away rapidly.

The *extent of the paralysis* may be of considerable value in differentiating hemorrhage from thrombosis. In the majority of cases of apoplexy from hemorrhage there is complete hemiplegia. It is decidedly unusual to meet with a monoplegia in hemorrhage. On the other hand thrombosis often affects the smaller cortical branches, producing a paralysis of limited extent or the loss of power appears first in one limb and later in another. Excluding hemorrhage the result of traumatism, a cortical lesion is nearly always thrombotic or embolic, so that if it can be established that the lesion is cortical it is far more likely to be due to thrombosis than to hemorrhage. In favor of a cortical lesion are the following points: the paralysis may be limited to the face, to the arm or leg, or if there is hemiplegia it is incomplete, one limb being much more affected than the other. Aphasia is also a symptom of a cortical lesion, as damage to the internal capsule will probably not produce complete aphasia. Hence, a true aphasia is much more frequent in thrombosis than in hemorrhage.

Reactionary Fever and Other Signs—On the first day of the attack in hemorrhage the temperature is usually subnormal, but this is followed by a rise to 101 to 103, occasionally higher. In thrombosis and embolism, as a rule, there is no initial fall and little or no subsequent rise in temperature. The pulse in hemorrhage is full, of high tension, slow and often irregular; in thrombosis it is normal or weak and of low tension; in embolism it is the pulse of the cardiac condition. The pupils in hemorrhage are often irregular and do not react to light; in thrombosis and embolism they are usually regular and react to light.

Rapidity of Recovery—In hemorrhage, recovery from the paralysis is slow and some permanent defect always remains. In thrombosis, and likewise in embolism, there is often marked improvement within the week, sometimes in the first two or three days, that is, as soon as the collateral circulation is established. Occasionally in thrombosis there is a marked return of power on the second day followed on the third day by an increase in the extent of the paralysis. This may be due to extension of the thrombus into other branches, but in many instances is the result of temporary vascular changes with œdema, the symptoms improving again in a few days.

Prognosis—I shall not attempt to discuss the various indications which may assist us in predicting the outcome of a case. An attack of apoplexy due to cerebral hemorrhage is much more likely to prove fatal than that due to thrombosis or embolism, in fact some of the English neurologists claim that the great majority of hemorrhagic apoplexies are fatal. We all recognize the grave import of deep and prolonged coma, of stertorous breathing, etc., and in cases presenting such symptoms we are rarely mistaken in predicting an unfavorable termination. But in cases where consciousness is not entirely lost, though hemiplegia may be complete, prognosis is so difficult that the only safe ground is to withhold an opinion and await developments. In thrombosis or embolism the collateral circulation may be quickly established and a remarkable improvement in all the symptoms may be seen even as early as the second day. On the other hand, the later development of œdema or extension of the thrombus in an apparently mild case may cause an aggravation of the symptoms with coma and a rapidly fatal termination. In thrombosis the third day often seems to be critical and probably coincides with the development of extensive œdema in and about the area of brain supplied by the occluded vessel. Again in thrombosis one must not forget the danger of recurrences. Even in syphilitic cases this is, I believe, an ever-present danger, for when cerebral syphilis has advanced to the apoplectic or paralytic stage, I have but little confidence in complete cures by even large doses of the iodides. It cannot be expected that these drugs will restore degenerated tissues to their normal condition.